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ORIGINAL ARTICLE

The relationship between blood pressure and blood lead in NHANES III

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There is no general agreement as to whether low-level lead exposure increases blood pressure. The present study examined the correlation between blood pressure and blood lead in the NHANES III database (1988–1994). Analyses were performed for all adults (⩾20 years), and reported separately for white males (*n* = 4685), white females (*n* = 5138), black males (*n* = 1761) and black females (*n* = 2197). Significant covariates of blood pressure were selected by stepwise regression. The change in blood pressure that would be associated with a doubling of blood lead was calculated from the adjusted regression coefficients. Mean systolic/diastolic blood pressure was 123/76 mm Hg in white males, 119/70 mm Hg in white females, 126/77 mm Hg in black males and 121/72 mm Hg in black females. Median blood lead was 174 nmol/L (3.6 μg/dL), 101 nmol/L (2.1 μg/dL), 203 nmol/L (4.2 μg/dL) and 111 nmol/L (2.3 μg/dL), respectively. For a doubling of blood lead, the changes in systolic blood pressure were 0.3 (95% confidence interval: −0.2 to 0.7, *P* = 0.29), 0.1 (−0.4 to 0.5, *P* = 0.80), 0.9 (0.04 to 1.8, *P* = 0.04) and 1.2 (0.4 to 2.0, *P* = 0.004) mm Hg, respectively and the changes in diastolic blood pressure were −0.6 (−0.9 to −0.3, *P* = 0.0003), −0.2 (−0.5 to −0.1, *P* = 0.13), 0.3 (−0.3 to 1.0, *P* = 0.28) and 0.5 (0.01 to 1.1, *P* = 0.047) mm Hg, respectively. In conclusion, there is no consistent relationship between blood pressure and blood lead in the NHANES III dataset.
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Keywords: blood pressure; lead; heavy metals

Introduction

The issue of whether low-level lead exposure results in elevated blood pressure is still a matter of scientific debate. Contradictory results have been published, even when the data from a single study were analysed by different investigators. For example, in their analysis of the second National Health and Nutrition Examination Survey (NHANES II), some authors reported a significant and positive association between blood lead and blood pressure.[1–3] Others, however, have questioned the selection criteria and statistical methods applied in these calculations and found that there was no consistent relation between blood lead and blood pressure.[4]

In a recent meta-analysis (23 studies involving 33 141 subjects), we reported that at low to moderate levels of lead exposure, a doubling of the blood lead concentration was associated with significant but modest increases in blood pressure, ie 1.0 mm Hg ($P = 0.002$) systolic and 0.6 mm Hg ($P = 0.02$) diastolic.[5] Another meta-analysis of 15 studies (total number of subjects not reported) found a significant increase of 1.2 mm Hg in systolic blood pressure when mean blood lead doubled.[6]

The mean blood lead levels in the general population (1–74 years) declined from 620 nmol/L (12.8 μg/dL) in NHANES II (1976–1980) to 140 nmol/L (2.9 μg/dL) in phase 1 of NHANES III (1988–1991)[7] and continued to decline in phase 2 of NHANES III (1991–1994).[8] The unresolved question of a possible lead-related increase of blood pressure, and the considerable change in blood lead levels over time, prompted our examination of the relation between blood lead and blood pressure, using NHANES III data.

Materials and methods

NHANES III, conducted from 1988 through 1994, was designed to provide national estimates of the health and nutritional status of the United States' civilian, non-institutionalised population aged 2 months and older. The NHANES III database was obtained on CD-ROM from the National Centre for Health Statistics. The sampling procedures, examination procedures, laboratory performance and quality control measures have been described.[9]

Blood pressure was measured at the MEC by trained physicians according to American Heart

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Association guidelines[10] and was the mean of three readings in the sitting position. Dietary calcium, sodium and potassium intake (mg/day) were estimated by a 24-h recall questionnaire. Diabetes was coded if either disease code (ICD-9) 250 was specified or if the subject self-reported to have diabetes. Diuretics, calcium channel blockers, carbonic anhydrase inhibitors, beta-blockers, alpha-agonists and angiotensin-converting enzyme (ACE) inhibitors were defined as antihypertensive drugs. The poverty index, ie a standardised income variable, was used as measure for socio-economic status. Since the poverty index had many missing values (1464 (9.5%) on a total sample of 15431), all analyses were done with and without inclusion of the poverty index. Lead in blood (nmol/L) was measured by atomic absorption spectrometry as described by Miller *et al*.[11] Serum total protein (g/L), serum total calcium (mmol/L) and haematocrit (L/L) were measured by standard procedures.[9]

Data management and statistical analysis was done with the SAS software, version 6.12. Subjects below 20 years and of races other than black or white were excluded from the analysis. This left us with a sample size of 18220 subjects, of whom 15431 had at least one valid blood pressure reading. Separate analyses were done for white males, white females, black males and black females. The weighting factors available in the dataset were used to correct for non-responder bias and over- or under-selection of certain age, gender or race-ethnicity groups. Data that were not normally distributed, as tested by a Kolmogorov-Smirnov test, were log-transformed and described by the median and interquartile range. In a first step of the analysis, we computed unadjusted regression coefficients between blood pressure and blood lead using single regression analysis. Effect sizes with 95% confidence intervals were calculated from the regression coefficients for a doubling of the blood lead concentration. As the association between blood pressure and blood lead may by confounded by other variables, in a second step of the analysis, significant covariables of blood pressure were identified by a stepwise regression analysis for the four race-gender groups separately. We made an *a priori* decision to include known confounders in the model regardless of significance, ie age, age^2, body-mass index, haematocrit, smoking, alcohol consumption and use of antihypertensive drugs. The other variables (coffee consumption, dietary calcium, dietary sodium/potassium ratio, serum total protein, serum total calcium, presence of diabetes and poverty index) required a 0.05 significance level to enter and to stay in the stepwise model. Finally, allowing for significant covariates, the relation between blood pressure and blood lead was determined by multiple regression analysis and effect sizes, with 95% confidence intervals, were calculated for a doubling of the blood lead concentration from the regression coefficients for blood lead in the multiple regression analysis.

Results

Mean systolic and diastolic blood pressures and blood lead levels were significantly higher in blacks as compared with whites and in males as compared with females (Table 1). Because blood pressure and most other characteristics differed according to race and gender (Table 1), single and multiple regression analyses were performed in four race-gender groups separately. A correlation matrix between systolic blood pressure, diastolic blood pressure, the blood lead concentration and all other variables is given in Table 2.

Without adjustment for confounders, systolic blood pressures was positively associated with blood lead concentration (Figure 1), while diastolic blood pressure showed a weakly positive association or no relation with the blood lead concentration (Figure 2). In single regression analysis, a doubling of the blood lead concentration was associated with increases in systolic blood pressure of 2.1 mm Hg (95% CI: 1.6 to 2.5 mm Hg; $P < 0.001$) in white males, 5.1 mm Hg (4.6 to 5.6 mm Hg; $P < 0.001$) in white females, 3.8 mm Hg (3.0 to 4.6 mm Hg; $P < 0.001$) in black males and 5.6 mm Hg (4.8 to 6.5 mm Hg; $P < 0.001$) mm Hg in black females. Diastolic blood pressure was not significantly correlated with blood lead in white males (–0.3 mm Hg (–0.6 to 0.1 mm Hg; $P = 0.08$) for a doubling of the blood lead concentration). However, in the other race-gender groups, diastolic blood pressure increased with elevated blood lead levels. For a doubling of the blood lead concentration, the estimates were 0.5 mm Hg (0.3 to 0.8 mm Hg; $P < 0.001$) in white females, 1.1 mm Hg (0.5 to 1.6 mm Hg; $P < 0.001$) in black males, and 1.4 mm Hg (0.9 to 1.9 mm Hg; $P < 0.001$) in black females.

In multiple regression analysis with adjustment for important or significant confounders, systolic blood pressure was positively and independently correlated with blood lead in black subjects, but not in white subjects (Table 3). The change in blood pressure that would be associated with a doubling of the blood lead concentration was 0.3 mm Hg (95% Cl: –0.2 to 0.7 mm Hg; $P = 0.29$) in white males, 0.1 mm Hg (–0.4 to 0.5 mm Hg; $P = 0.80$) in white females, 0.9 mm Hg (0.04 to 1.8 mm Hg; $P = 0.04$) in black males and 1.2 mm Hg (0.4 to 2.0 mm Hg; $P = 0.004$) in black females (Figure 3). The positive relationship between systolic blood pressure and blood lead in black males weakened and became non-significant when the poverty index was included as an independent covariable in the model. The effect size associated with a doubling of the blood lead concentration then was 0.6 (–0.3 to 1.5; $P = 0.23$; $n = 1619$).

After adjustment for covariates in multiple regression analysis, the relation between diastolic

Table 1 Descriptive statistics of variables used in the regression analysis for four race-gender groups

	White males	*White females*	*Black males*	*Black females*	*P for race*	*P for gender*
n	4685	5138	1761	2197		
Dependent variables in regression analysis						
Systolic blood pressure (mm Hg)	123.0 (16.1)	118.6 (20.3)	125.8 (17.6)	121.1 (22.0)	<0.001	<0.001
Diastolic blood pressure (mm Hg)	75.7 (10.4)	70.1 (10.1)	77.5 (11.9)	72.1 (12.3)	<0.001	<0.001
Independent variables in regression analysis						
Blood lead						
(nmol/L)	174 (111–256)	101 (63–164)	203 (130–314)	111 (68–188)	<0.001	<0.001
(μg/dL)	3.6 (2.3–5.3)	2.1 (1.3–3.4)	4.2 (2.7–6.5)	2.3 (1.4–3.9)		
Age (years)	44.3 (16.6)	46.2 (17.8)	40.5 (15.3)	41.5 (16.3)	<0.001	<0.001
Body-mass index (kg/m^2)	26.7 (4.8)	26.2 (6.1)	26.6 (5.4)	28.7 (7.1)	<0.001	0.03
% smokers	34.1%	25.2%	41.7%	27.3%	<0.001	<0.001
% alcohol consumers	32.0%	18.2%	34.6%	15.4%	0.04	<0.001
% subjects drinking coffee	73.1%	64.4%	52.2%	48.5%	<0.001	<0.001
Dietary calcium (mg/day)	884 (551–1300)	637 (411–964)	631 (378–982)	460 (287–730)	<0.001	<0.001
Dietary sodium/potassium ratio	1.23 (0.91–1.63)	1.13 (0.82–1.56)	1.45 (1.08–1.98)	1.42 (1.00–1.91)	<0.001	<0.001
Haematocrit (L/L)	0.45 (0.03)	0.40 (0.03)	0.44 (0.03)	0.38 (0.03)	<0.001	<0.001
Serum total calcium (mmol/L)	2.33 (0.10)	2.31 (0.11)	2.34 (0.10)	2.31 (0.11)	0.22	<0.001
Serum total protein (g/L)	73.3 (4.3)	72.1 (4.3)	75.9 (4.8)	74.8 (4.8)	<0.001	<0.001
Poverty index*	3.38 (1.92)	3.16 (1.89)	2.23 (1.58)	1.91 (1.45)	<0.001	<0.001
% subjects using antihypertensive drugs	13.7%	17.8%	13.9%	20.6%	<0.001	<0.001
% subjects with diabetes	5.1%	5.5%	5.5%	9.1%	<0.001	<0.001

Data that are normally distributed are presented as means (standard deviation); data that are not normally distributed are presented as median (interquartile range); frequencies are given as percentage.
*n = 4274 in white males; n = 4670 in white females; n = 1619 in black males; n = 1961 in black females.

Table 2 Correlation matrix between systolic blood pressure (SBP), diastolic blood pressure (DBP), blood lead concentration (BPb) and all other variables used in the regression analysis for four race-gender groups

	White males			*White females*			*Black males*			*Black females*		
	SBP	*DBP*	*BPb**	*SBP*	*DBP*	*BPb**	*SBP*	*DBP*	*BPb**	*SBP*	*DBP*	*BPb**
DBP	0.46^c	–	–0.04^a	0.44^c	–	0.05^c	0.50^c	–	0.09^c	0.57^c	–	0.11^c
BPb*	0.11^c	–0.04^a	–	0.25^c	0.05^c	–	0.20^c	0.09^c	–	0.26^c	0.11^c	–
Age	0.45^c	0.04^b	0.22^c	0.63^c	0.13^c	0.42^c	0.44^c	0.15^c	0.40^c	0.57^c	0.18^c	0.39^c
Body-mass index	0.20^c	0.26^c	–0.06^c	0.26^c	0.31^c	0.02	0.19^c	0.16^c	–0.09^c	0.21^c	0.19^c	–0.05^a
% smokers†	–0.12^c	–0.06^c	0.19^c	–0.17^c	–0.09^c	0.11^c	–0.02	–0.06^a	0.30^c	–0.07^c	–0.06^b	0.26^c
% drinking alcohol†	–0.02	0.04^b	0.11^c	–0.05^a	0.01	0.05^c	–0.03	–0.01	0.10^c	–0.06^b	0.01	0.11^c
% drinking coffee†	0.03^a	0.03^a	0.10^c	0.04^a	0.01	0.13^c	0.03	–0.02	0.15^c	0.10^c	0.06^b	0.11^c
Dietary calcium*	–0.10^c	–0.01	–0.16^c	–0.08^c	–0.05^b	–0.12^c	–0.11^c	–0.10^c	–0.18^c	–0.09^c	–0.07^b	–0.09^c
Dietary Na/K ratio*	–0.05^b	0.02	–0.04^b	–0.12^c	–0.01	–0.15^c	–0.04	–0.01	0.03	–0.10^c	0.04	–0.08^c
Haematocrit	–0.07^c	0.14^c	0.02	–0.09^c	0.16^c	0.19^c	–0.09^c	0.10^c	–0.05^a	0.06^b	0.10^c	0.16^c
Serum total calcium	0.03	0.03^a	–0.01	0.14^c	0.06^c	–0.14^c	–0.02	0.02	–0.03	0.13^c	0.02	0.13^c
Serum total protein	0.02	0.05^b	–0.04^a	0.04^b	0.10^c	–0.02	0.02	0.08^c	–0.01	0.11^c	0.14^c	0.02
% on AH drugs†	0.30^c	0.04^b	0.04^b	0.42^c	0.10^c	0.15^c	0.28^c	0.13^c	0.16^c	0.10^c	0.06^b	0.11^c
% with diabetes†	0.13^b	–0.02	–0.04^b	0.04^a	0.01	0.13^c	0.14^c	–0.02	0.04	0.19^c	0.05^a	0.07^c

$^aP < 0.05$; $^bP < 0.01$; $^cP < 0.001$. *Logarithmically transformed.
Pearson correlation coefficient for continuous variables; †Spearman correlation coefficients for dichotomous variables.

blood pressure and blood lead was significant and inverse in white males, not significant in white females or black males, and of borderline significance and positive in black females (Table 4). The effect sizes for a doubling of blood lead were –0.6 mm Hg (95% Cl: –0.9 to –0.3; $P < 0.001$) in white males, –0.2 mm Hg (–0.5 to 0.1; $P = 0.13$) in white females, 0.3 mm Hg (–0.3 to 1.0; $P = 0.28$) in black males and 0.5 mm Hg (0.01 to 1.1; $P = 0.047$) in black females (Figure 3).

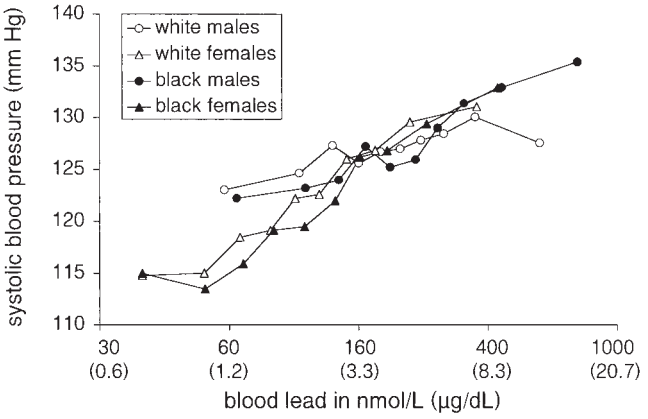

Figure 1 Unadjusted relation between systolic blood pressure and blood lead. Mean blood pressure is given per decile of blood lead in four race-gender groups.

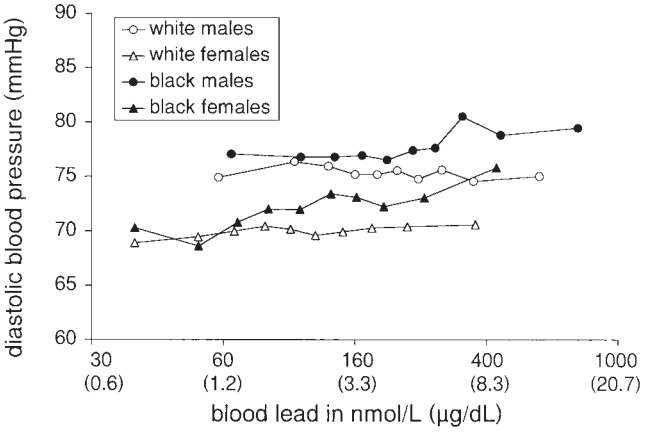

Figure 2 Unadjusted relation between diastolic blood pressure and blood lead. Mean blood pressure is given per decile of blood lead in four race-gender groups.

Discussion

We used NHANES III data to examine the relationship between blood pressure and blood lead. A significant and positive relationship was found between systolic blood pressure and blood lead, but only in blacks. The relation between diastolic blood pressure and blood lead was inconsistent for the different race-gender groups.

Several authors[1,2,4] studied the relationship between blood pressure and blood lead in NHANES II and reported for a doubling of blood lead a change in blood pressure ranging from non-significant to a maximum increase of 5.8 mm Hg ($P < 0.001$) systolic and 2.7 mm Hg ($P < 0.001$) diastolic. These contradictory results probably are due to variation in criteria for including participants in the analysis (age groups, gender, race), weighting factors, covariates selected as confounders and statistical methods. Similarly, in the British Regional Heart Study (BRHS) inconsistent findings depending on statistical methods and use of covariables were reported.[12,13] The researcher concluded that for a doubling of blood lead, there was a weak but significant increase of 1.45 mm Hg in systolic blood pressure.[13]

Blood lead levels in Western countries have dropped considerably throughout the last decades due to the reduction of lead in gasoline and in soldered food and drink cans.[7,14–16] In the general US population (1–74 years) mean blood lead levels dropped from 620 nmol/L (12.8 µg/dL) in NHANES

Table 3 Covariates of systolic blood pressure in multiple regression analysis by race and sex

	White males	*White females*	*Black males*	*Black females*
n	4685	5138	1761	2197
R^2	0.26	0.47	0.25	0.38
Intercept	58.1[c]	66.2[c]	80.2[c]	60.4[c]
Independent variables				
Blood lead*	0.84	0.21	2.98[a]	3.93[b]
Age	−0.10	−0.16[a]	−0.04	0.74[c]
Age^2	0.005[c]	0.008[c]	0.005	−0.001
Body-mass index	0.56[c]	0.59[c]	0.59[c]	0.37[c]
Haematocrit	9.89	−5.13	11.30	−23.91[a]
Smoking	0.18	−1.88[c]	−0.14	−0.35
Alcohol consumption	1.96[c]	2.13[c]	1.37	2.38[a]
Antihypertensive drugs	4.46[c]	6.22[c]	5.46[c]	9.10[c]
Coffee consumption	–	–	–	–
Dietary calcium*	−1.69[a]	–	–	–
Dietary sodium/potassium ratio*	3.05[b]	–	–	4.74[b]
Serum total protein	2.59[c]	3.65[c]	2.36[b]	4.22[c]
Serum total calcium	9.99[c]	–	–	–
Presence of diabetes	–	3.17[c]	–	–

Blood lead, age, age^2, body-mass index, haematocrit, smoking, alcohol consumption and intake of antihypertensive drugs were forced in the regression model. Other covariates were selected by a stepwise regression procedure with a significance level of 0.05 to enter and to stay in the model.
[a]$P < 0.05$; [b]$P < 0.01$; [c]$P < 0.001$. *Logarithmically transformed.

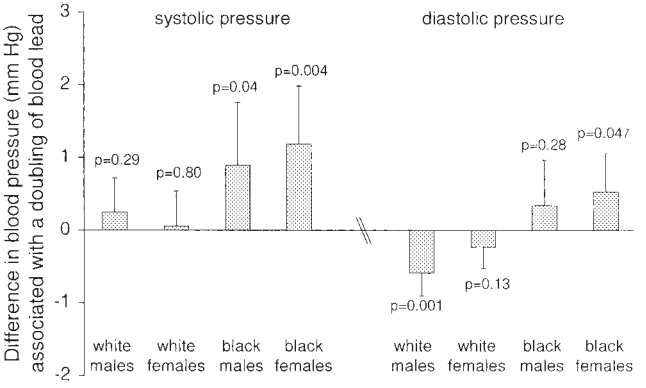

Figure 3 Adjusted differences in systolic and diastolic blood pressure associated with a doubling of the blood lead concentrations in four race-gender groups. Estimates (± s.e.) were calculated from regression coefficients adjusted for the covariates listed in Table 2 (systolic blood pressure) and Table 3 (diastolic blood pressure).

Table 4 Covariates of diastolic blood pressure in multiple regression analysis by race and sex

	White males	*White females*	*Black males*	*Black females*
n	4681	5126	1760	2186
R^2	0.15	0.17	0.13	0.16
Intercept	–2.6	16.9[c]	16.0[a]	21.5[b]
Independent variables				
Blood lead*	–1.95[c]	–0.76	1.13	1.78[a]
Age	0.87[c]	0.55[c]	1.09[c]	0.94[c]
Age^2	–0.008[c]	–0.005[c]	–0.01[c]	–0.009[c]
Body-mass index	0.46[c]	0.43[c]	0.26[c]	0.20[c]
Haematocrit	50.0[c]	42.9[c]	47.1[c]	28.5[c]
Smoking	–1.18[c]	–2.29[c]	–1.62[b]	–1.95[b]
Alcohol consumption	1.38[c]	0.61	0.17	2.61[c]
Antihypertensive drugs	1.31[b]	1.54[c]	2.79[b]	4.02[c]
Coffee consumption	–	–	–1.30[a]	–
Dietary calcium*	–	–0.93[a]	–1.89[a]	–
Dietary sodium/potassium ratio*	1.83[a]	–	–	4.89[c]
Serum total protein	0.96[b]	1.97[c]	2.16[c]	3.50[c]
Serum total calcium	6.50[c]	–	–	–5.58[a]
Presence of diabetes	–2.71[c]	–4.10[c]	–4.53[c]	

Blood lead, age, age^2, body-mass index, haematocrit, smoking, alcohol consumption and intake of antihypertensive drugs were forced in the regression model. Other covariates were selected by a stepwise regression procedure with a significance level of 0.05 to enter and to stay in the model.
[a]$P < 0.05$; [b]$P < 0.01$; [c]$P < 0.001$. *Logarithmically transformed.

II (1976–1980) to 140 nmol/L (2.9 μg/dL) in the first phase of NHANES III (1988–1991).[7] In Belgium, mean blood lead levels in a random population sample ($n = 728$; 49% men; age range: 20–82 years) averaged 420 nmol/L (8.7 μg/dL) in the period 1985–1989. After a median follow-up of 5.2-years, blood lead levels had dropped by 32% to a mean value of 140 nmol/L (2.9 μg/dL).[16] In this prospective population study, no consistent association was found between the change in blood pressure and blood lead. In addition, the blood lead (or zinc protoporphyrin) concentration at baseline did not predict the development of hypertension in the follow-up period.[16] These conclusions are in agreement with the current findings in NHANES III, ie there is no significant relationship between blood lead levels and blood pressure in white males or females. Also, in a population with high industrial exposure levels (mean blood lead levels: 1525 nmol/L (31.6 μg/dL)), no significant relation was found between blood pressure and blood lead after adjustment for confounders.[17]

Unlike in white subjects, a significant and positive association was found between blood pressure and blood lead in black subjects. This is in agreement with a prospective study performed in San Francisco bus drivers. Systolic and diastolic blood pressure increased significantly with rising blood lead levels in blacks, but not in whites.[18] Also in NHANES II, race was an important covariable in the analysis of blood lead and blood pressure.[19]

Blood lead levels are usually higher in blacks than in whites. This is often attributed to socio-economic factors, eg to a higher degree of urbanisation, higher occupational exposure or housing in older buildings with lead-containing paint. In NHANES II, however, black/white differences could not be explained by demographic or socio-economic factors, since race differences were found in all age groups, in rural residents and urban dwellers, and in families with low, moderate and high incomes.[20]

It is well documented that blood pressure and mortality from hypertensive disease are higher in blacks than in whites.[21,22] The reasons for these racial differences remain to be determined. Differences in food consumption patterns (higher sodium, lower potassium and calcium intake), higher prevalence of obesity, lower socio-economic status (resulting for example in less access to antihypertensive treatment) may contribute to the higher blood pressure in blacks.[23,24] Genetic differences also may be the cause of a higher inborn susceptibility to hypertension in blacks. Candidate susceptibility genes remain the source of speculation, but differences in sodium sensitivity, cation transport, vascular injury, insulin resistance or adrenergic activity have been postulated as being important.[23,25] In the present analysis, blood pressure was corrected for body-mass index, dietary calcium, dietary sodium, dietary potassium, poverty index and the use of antihypertensive drugs, but the relation between blood pressure and blood lead remained significant in blacks. Though blood lead levels were lower in whites, the overlap in blood lead levels between blacks and whites was large, so that it seems unlikely that there is a threshold value for an effect of lead on blood pressure that was exceeded in blacks but not in whites. Most likely, there is still unidentified confounding in blacks that is not taken into account in our calculations.

In conclusion, there is no consistent relationship between blood pressure and blood lead in NHANES III.

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